# INSECTS AND INFECTION.*

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You will pardon me if I explain that the title of this long paper was chosen for the sake of brevity. I shall speak not only of the role of the *Insecta* in the transmission of infectious agents but also of other *Arthropoda* such as some of the *Arachnoidea,* e. g., the ticks.

Our ideas concerning the hygiene of infectious processes occurring in man and other animals have undergone gradual change. One might consume several hours in simply reading off the names of those who have contributed to this change. While much of the advance made in recent years is to be credited to the medical profession, they in turn were often greatly influenced by those working along strictly biological lines. This is especially true of the development of our knowledge concerning the life history of animal parasites in general. As I shall hope to show, human medicine always must be grateful to workers in general biology and comparative pathology for many fundamental conceptions.

The idea that insects might transmit infectious diseases from one animal to another, dates back many years, and many observers had speculated upon its possibility. But it was one of our own countrymen who first demonstrated to the world the actual role played by a haemophagous parasite in the transmission of a diasease. I refer to the remarkable work of Dr. Theobald Smith on Texas fever in cattle.

This noted comparative pathologist worked for three years on the transmission of this disease before publishing his observations. He was so fearful lest his experiments, which had led him to a hitherto unheard of conclusion, might be faulty, that he repeated them over and over and only when repeated confirmatory results were obtained did he give them publicity. The eludication of the mode of transmission in Texas fever is all the more remarkable when one reflects that the disease is not transmitted by the tick which sucks up infected blood but by her progeny; for having sucked blood, the tick drops off her host, lays her eggs, and dies.

Since Pasteur in 1870 demonstrated the "pebrine bodies" in the silk worm disease of France, it has been known that an insect may lay infected eggs and so hand down a disease to its offspring.(1) In this instance Pasteur was dealing with a disease among insects transmissible through ingesta, traumatic injuries inflicted upon one another and by heredity. Infected eggs could be recognized under the microscope and Pasteur showed that the little caterpillars emerging from the infected eggs must be destroyed in order to prevent their infecting healthy larvae. This discovery was fundamental; and while it meant millions of francs to the people of France, it meant more to the world—for it was a truly dramatic demonstration of the value of a hygiene based on facts obtained through patient research.

The work of Dr. Theobald Smith was equally fundamental in character in that it presented to the scientific world the first definitely proven instance of the transmission of a parasite from one mammal to another by one of the arthropoda. The parasite producing Texas fever is so small and its wanderings through its definitive host so complicated that until perhaps recently, the stages of its extra-corporeal existence remained unknown.(2)

Some work done not long ago by Dr. Fritz Schaudinn might instance a series of metamorphoses which probably rival those of the piroplasma in their complexity. Celli and Sanfelice (1891) had described the crescentic forms of a parasite occurring in the blood of a little owl (*Athenae noctuae*) under the name of *Haemoproteus noctuae* (=Haeteridium). Schaudinn thought he had demonstrated that this parasite was in reality a trypanosome which maintained its existence by an alternation of generative processes and a change of hosts.(2)

He not only followed the asexual life history of the *"Trypanosoma noctuae"* within the blood of the owl which acts as one of its intermediate hosts, but step by step, as he thought, cleared up the complicated nature of its extra-corporeal existence within the stomach and tissues of a mosquito (*Culex pipiens*) which acts as its definitive host.

To me, the most interesting discovery made by him does not concern the details of the metamorphosis of the parasite within the insect or the fact that after feeding on infected blood it can reinoculate the disease. Analogous instances had been furnished long before (1898) by Ross in the case of Proteosoma infection of birds; and by Manson and his son, in the case of human malaria.

The most interesting part of Schaudinn's work lies in the fact that certain phases of this trypanosome may also pass through the eggs and larvae into the next generation of perfect mosquitoes. And further, that the insect which has just emerged from its pupa can not transmit the disease at once, but can at the time of its third feeding! However, it might be well to state here that the recent work by Novy and his associates on the *natural* occurrence of trypanosomes in birds and mosquitoes has thrown doubt on the validity of Schaudinn's observations.

It is practically impossible to trace the origin of conceptions which have led to fruitful results in science but it seems quite possible that that patient missionary physician, David Livingstone, may have influenced some of the early workers by his reports (1857) of the existence of a fatal epidemic disease among animals in the wilds of Africa. This disease was popularly believed by the negroes to be transmitted by biting flies and was known as "Tsetze-fly Disease."

And again, a missionary physician in China, Dr. Manson, between 1879 and 1883, observed that the embryos of *Filaria bancrofti* were taken up by a cer-

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tain species of mosquito and underwent a further development in its body. At this time he believed that the developed embryo might gain access to water and through this medium return again to a human host. Then came the brilliant inductions of Dr. Manson (1896) concerning the life history of the malarial parasite. He was, no doubt, influenced in his reasoning by his previous work on the development of *Filaria nocturna* in mosquitoes, and, probably too, by the work of Dr. Theobald Smith on Texas fever. He reasoned *a priori*:

(1) Since the parasite is encased in the blood cells and can not escape from one host to another by its own efforts, it must be removed by some blood-sucking animal—probably a suctorial insect; an insect common in swampy places where malaria is common—hence probably a mosquito.

(2) That the flagellated forms developing in malarial blood exposed to the air for a few minutes were not degenerated forms but represented the extra-corporeal homologue of the intra-corporeal spore.[3]

It is interesting to note here that for a long time previous to this the inhabitants of the malarial districts of Italy had believed that the mosquio played some role in the transmission of the disease, and according to Koch, this same belief was shared by the natives of Africa.

Manson's first hypothesis was confirmed by Ross in 1897, when he described the growth of pigmented parasites in the body of an Anopheles fed on aestivo-autumnal blood. His subsequent observations on the metamorphosis of the *Proteosoma* of birds and the subsequent researches of Grassi and many other investigators have amply confirmed this hypothesis.

The second hypothesis, in regard to the flagellated forms, received its first support from this country. The flagellated form was considered by Danilewsky to be an independent parasite and he named it *Polymitus*. While Manson, Laveran and Metchnikoff believed it was connected with the further development of the parasites of malaria, Grassi, Celli, Felletti, Sanfelice and others believed that they were degenerated forms of the parasites produced by exposure to a lower temperature than normal. In 1897, Opie noted that there were two kinds of spheres in the malaria of birds—coarsely granular and hyaline forms, only the latter of which developed polymitus forms. At the same time MacCallum made the important discovery, while studying the halteridium of crows, that these flagellated forms were actually *sexual forms*.

Since then our knowledge of the role of insects in the transmission of infectious diseases has taken rapid strides and physicians today must have at least a literary knowledge of the subject if they pretend to qualify in the Science of Hygiene.

We can logically divide the subject matter into three parts:

(1) Insecta, which act as intermediate hosts for parasites.

(2) Insecta and Arachnoidea which act as definitive hosts for parasites.

(3) Insecta and Arachnoidea by which parasites are transplanted mechanically.

I. *Insecta which acts as intermediate hosts for parasites*—The early observations of Manson on the metamorphosis of *Filaria nocturna* in the bodies of certain mosquitoes (*Culex fatigans*) were confirmed and extended by the work of Low in 1900. His discovery of the developed embryos in the mouth-parts of this insect led to the generally accepted theory that the parasites are reinoculated into their definitive host by the bite of certain mosquitoes. This opinion received strong support from the work of Grassi and Noe on the *Filaria immitis* of dogs. These workers were able to transmit the parasites to healthy dogs through the bite of infected mosquitoes (*Culex pipiens*). James showed that in India, both Anopheles and Culex may act as intermediate hosts for *Filaria nocturna* and the work of Grassi and Noe proves the same lack of specific selectivity in the case of *Filaria immitis*.

However, the work of Dr. Low seems to indicate that the *Filaria perstans* is more particular in its choice of an intermediate host, for out of a large number of careful experiments with many different species and genera of mosquitoes, only one *Taeniorhynchus fuscopennatus* was found suitable for its metamorphosis.

If we consider, for a moment, another form of infection in dogs, due to the *Filaria recondita,* we find that Grassi found its intermediate host to be the dog flea.

In the case of the Guinea of Medina worm, it is altogether probable from the work of Fedschenko in Turkestan—since confirmed by Manson and Blanchard—that some crustacean, like the fresh water cyclops, acts as intermediate host.

II. *Insecta and Arachnoidea which act as definitive hosts for parasites*—I have spoken of the transmission of Texas fever by the *Rhipicephalus annulatus*. In Africa this form of piroplasmosis, known as Rhodesian or Red Water fever, is transmited by the *Rhipicephalus appendiculatus*. In Germany it is transmitted by the *Ixodes ricinus*. I will merely mention the piroplasmosis of sheep and dogs transmitted by the ticks *Amblyomma hebraeum* and *Haemaphysalis Leachi* respectively.

Then I would recall the recent work of Dr. H. T. Ricketts who has shown that the spotted fever of Montana, and neighboring states, may be transmitted from one experimental animal to another by a tick (*Dermacentor occidentalis*). The seasonal occurrence of this disease would seem to indicate that its virus finds a definitive host in the tick or in some mammal other than man.

I might mention here the role of *Culex pipiens* acting as a definitive host for the *Trypanosoma noctuae* as described by Schudinn, but would remind you of the doubt thrown upon his observations by the work of Novy and his associates.

Under this heading we may also include the *Anophelenae* and the *Stegomyia Calopus* which, as

you all well know, act as the definitive hosts for the parasites of malaria and yellow fever. There is no evidence that malaria may be transmitted by heredity through the Anopheles. So far as I know there is only one such instance on record in the case of yellow fever. This is reported by Marchoux and Simon[4] from Brazil and must be open to question since other investigators have been unable to confirm it.

III. *Insecta and Arachnoidea by which parasites are transplanted mechanically*—Ever since the early experimental work of Simond Hankin and Nuttall, many attempts have been made to prove or disprove the role of the flea in the transmission of Bubonic plague. The frequent association between epidemic plague in rats and in man naturally suggested the possible existence of an insect intermediary. This association seems to have existed even in the earliest historical times. The Biblical account of an epidemic[5] would seem to show that the relation between mice and rats and epidemic buboes was recognized as early as 1141 B. C.

In the summer of 1905 I had the pleasure of visiting the plague research laboratories in Bombay. Plague was at its height that summer, reaching a mortality of 60,000 per week. The English Plague Commission, headed by Dr. Martin of the Lister Institute of Preventive Medicine, was then actively investigating the role played by the flea in the transmission of plague. I wish I could give you a picture of the enthusiasm with which these young and old men engaged upon their research, in the face of that awful melting heat. Reports of their extensive experiments furnish overwhelming proof that the flea is an important transmitting agent.

I would next speak of a group of diseases in which it is impossible to say at present whether the Arachnoidea concerned in their transmission act mechanically or as intermediate or definitive hosts—though it is probable that their action is mechanical: The recent studies of Novy and Knapp[6] and others on relapsing fever have thrown much doubt on the protozoan nature of many of the spiral organisms which Schaudinn classed as *spirochetes* and have proven, almost conclusively, that the *Spirillum Obermeieri* is to be classed with the bacteria. A number of these spirillar diseases in animals and man are transmitted by blood-sucking parasites. The spirillosis of fowls by the *Argas miniatus* (Marchoux and Salimbeni; Porrel and Marchoux); the cause of bovine spirillosis (*Spirillum Theileri*) by the *Rhipicephalus decoloratus.*

Sixteen years ago Pasternazki found the *Spirillum Obermeieri* viable after some time in the body of the leech. In 1902, Karlinski reported them viable in bedbugs thirty days after feeding on infected blood. From 1904 up to the present, through the work of Ross, Milne, Dutton, Todd, Koch, Novy, Knapp and others, the African "Tick Fever" has been proven to be a spirillosis showing slight variations from that discovered by Obermeier. It is transmitted by the bite of a tick (*Ornithodorus Savigni moubata*), and may be transmitted through the female and her eggs to her young. Neither Borrel, Marchoux, Dutton, Todd nor Koch have noted any developmental stages inside of these ticks and the work of Novy and Knapp seems to furnish justification for the belief that in these diseases we have bacteria which may be transmitted by heredity to the young of infected ticks.

We will now consider the mechanical action of various biting flies and fleas in the transplantation of parasites. The discovery of trypanosemes in the blood of rats was made by Lewis in 1879. In India, this was followed by their detection in the blood of horses suffering from "surra," by Evans, in 1880. Then came the discovery by Bruce (1894) that the dreaded Tsetze-fly disease of nagana of Africa was caused by a trypanosome. In 1898, Nepveu, in Algiers, described trypanosomes found in eight human beings, but his discovery was overlooked until Ford and Dutton, in 1901, discovered them in the blood of a patient in the River Gambia Colony in West Africa.

The popular conception that nagana was spread through the agency of Tsetze-flies was confirmed by the experimental work of Bruce, who showed that the *Glossina morsitans* was capable of inoculating the parasites immediately and up to within forty-eight hours after feeding on the blood of an infected animal. Since then the experimental work of Rodgers and Schilling in India; Voges in South America; Dutton, Brumpt and Koch in Africa; Curry, Musgrave and Clegg in the Philippines, and many others, have proven conclusively that various Tsetze-flies (Glossina), Gad flies (Tabanidæ) and stinging flies (Stomoxys) act as the chief transmitters of mammalian trypanosomiasis. Musgrave and Clegg also furnished experimental proof that the flea may transmit surra from dog to dog, rat to rat, and rat to dog. The flea had already been condemned as the transmitting agent in rat trypanosomiasis by the work of Plimmer, Bradford, Rabinowitch and Kempner. It is generally believed that the trypanosomes are carried in the simplest mechanical way by these insects. Koch, working in German East Africa, thought he could trace developmental forms in three species of Glossina (*morsitans, pallidipes, fusca*). However, the recent work of Novy in Michigan has upset and thrown much doubt upon the validity of Koch's observations by pointing out the necessity of distinguishing between *pathogenic* forms which may be taken up by flies and mosquitoes and those *non-pathogenic* forms which probably normally occur in many of these insects.

Leaving out of consideration the occasional wholesale dissemination of typhoid bacilli by a contaminated water or milk supply, their transference by contact and the measures to be taken in destroying these parasites as they leave an infected host during the disease and after convalescence, I wish to direct your attention to their mechanical transplantation by the various species of house flies (*Musca domestica, Anthomyia canicularis, Lucilia Caesar.*)

The important part played by these insects in the

dissemination of typhoid bacilli was shown by Majors Reed, Vaughan and Shakespeare in their report on the "Origin and Spread of Typhoid Fever in the United States Military Camps During the Spanish-American War in 1898." They note that "flies alternately visited and fed on the infected fecal matter and the food in the mess tents. More than once it happened when lime had been scattered over the fecal matter in the pits, flies with their feet covered with lime were seen walking over the food. Typhoid fever was much less frequent among members of messes who had their mess tents screened than among those who took no such precaution."

This report received valuable experimental support from the work of Dr. Alice Hamilton, carried on during the typhoid epidemic of Chicago in July, August and September of 1902. A number of workers had shown that flies walking over typhoid cultures picked up the germs and transplanted them shortly afterwards. But it remained for Dr. Hamilton to demonstrate[7] that flies caught in undrained privies, on the fences of yards, on the walls of houses, and in the room of a typhoid patient *actually carried* typhoid bacilli. Flies caught in such localities were used to inoculate 18 culture tubes and from 5 of these the typhoid bacillus was isolated.

No doubt the so-called "summer diarrhœa" of children, institutional and acute epidemic dysentery, with which the *bacillus dysenteriae* of Shiga, and related types of bacilli are associated, may be transferred in the same way.

There have been more than two millions of victims to cholera in the past six years. In this disease as in typhoid, man acts as chief carrier. Comparatively recent research has shown that apparently *healthy* individuals may act as carriers for these micro-organisms. The Germans style such individuals *Bacillen-traeger*. It is a well-known fact that patients recovering from typhoid often harbor and excrete typhoid bacilli in their urine for weeks; and the classical case of the bakeress of Strassburg, reported by Kayser, would seem to show that certain individuals may harbor these parasites in their gall bladder and excrete them in their feces for years.

In 1905, Gotschich found typical cholera spirilla in the intestines of pilgrims returned to Tor from Mecca. Cholera had failed to appear in Mecca that year and yet these perfectly healthy individuals had picked up and carried the germs for a period of more than five months. The specific identity of the germs isolated can hardly be questioned since they were also studied by Gaffky, Kohler, Kolle and Meinicke. The propagation of cholera from town to town and from one country to another is, no doubt, through human agency, but in its transfer from individual to individual, flies play an important role. In a disease like Asiatic cholera, where, in the acute stages, every drop of the intestinal evacuations contains literally thousands of spirilla, the chances of their successful transfer by flies can hardly be questioned. This belief is strongly supported by the classic experiments of Hankin in India, who found cholera spirilla in sterilized milk exposed to flies in an endemic focus.

Then, I have no doubt that flies play an important part in the spread of certain forms of ophthalmia, especially of Egyptian ophthalmia, or what is vulgarly known as "pink eye" in this country. In India I have seen dozens of flies crawling over the purulent ocular secretions of a child afflicted with this disease, and when brushed off, to fly away and alight on the dirty faces and eyes of neighboring children.

Those of us who know enough protect our children from the milk of tuberculous cattle. How well, let me ask you, do we protect their food stuffs against the fly? I would remind you of the interesting observations of Lord[8] who found that tubercle bacilli, taken up from sputum, undergo a marked proliferation in the intestinal tract of the common house fly and are discharged with its feces. The "specks" of such infested flies contained as many as 5000 bacilli; and according to Lord, thirty infected flies may deposit from 6 to 10 million tubercle bacilli in three days! Please compare these with the comparatively few bacilli to be found in milk of tuberculous origin.

According to L. O. Howard, extended observations have shown that over 99 per cent of the flies found in kitchens and dining-rooms and attracted to food supplies are house flies (*M. domestica*). Then, there are little fruit flies of the genus Drosophila, which you have all seen. Their life cycle is no doubt often represented by the vicious circle—eggs laid on over ripe fruit on a dining-room table, taken into the human alimentary tract, passed out with the feces, from which the insects hatch and fly to fruit on some one else's dining-room table. I was accustomed, during my youth in India, to see our food stuffs on and off the table protected by wire screens. It seems that local boards of health could do much to educate the ignorant of the necessity of protecting such food stuffs as milk, butter, bread, cold meats, fruit, etc., from flies.

Chantemesse quotes from an American writer: "It should be more of a disgrace for a house-keeper to have flies in her house than bed bugs in her beds;" but if you will reflect for a moment, many a poor house-keeper is not so culpable as many a board of health, which, year after year, allows piles of horse manure to lie unscreened and so donates to the public an annual visit from one of the plagues of Egypt.

It seems to me that in such a State as California—where such a great variation in topographical and climatic conditions obtain—many problems, connected with the transmission of infectious diseases by hæmophagous parasites, are open to study. We have had plague here; malaria and filariasis have been imported, and may be endemic in some sections.

Certainly there are sections in which diseases, if imported from tropical zones, could flourish.

May I ask what has been done to determine the

*presence, bionomics* and *distribution* of such insects as might play a role in their transmission?

(1) In 1884 Balbiani classified these "pebrine bodies" as microsporidia and now the parasite is known as Nosema Bombycis.

(2) R. Koch is said to have pictured his observations on its development in three varieties of ticks in East Africa (1905).

(2) By an alternation of generative processes is meant the alternation of a sexual with an asexual method of reproduction. This method of reproduction was first noted by Steentsrup in 1842, but probably the first biologist to properly interpret the significance of conjugation among the protozoa was Balbiani who in 1876 observed that a continual asexual division of certain forms resulted in decreased size and a general "lowering of the life energy." He rightly concluded that the purpose of conjugation was to rejuvenate the species. (Calkins.)

(3) Along with the publication of Manson's inductions came the exactly opposite opinions of Bignami who believed that the mosquitoes derived their infection from some as yet unknown stage of the malarial parasite occurring out in nature and subsequently inoculated man with their bite. He believed this opinion was supported in part by the role played by ticks in the transmission of Texas fever and also by the blood inoculation experiments of Gerhardt. But experiments conducted by Bignami and Dionisi failed to substantiate their views. Koch believed that there was truth in both views and further cited the analogy in the transmission of the Tsetze-fly disease—already established by the experimental work of Bruce.

(4) Ann. l'Inst. Past. 1906, xx, no. 1.

(5) 1st Sam., v. and vi.

(6) J. of Inf. Dis. 1906, 3, 291.

(7) Jour. A. M. A., 1903, Feb. 28th.

(8) Pub. of the Mass. Gen. Hosp., 1906, 1, 118.

## BRAIN SYMPTOMS OF TYPHOID FEVER SIMULATING THOSE OF MASTOIDITIS.*

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Dr. E. W. Day and Chevalier Jackson in a very thorough paper (Laryngoscope, 1904, Vol. XIV), drew our attention to the fact that ear affections are much more frequent in typhoid fever than are usually supposed. This can easily be explained by the fact that a routine examination of the ears of typhoid patients is made in very few hospitals and hardly ever in private practice. In over 800 cases of typhoid fever they found 88 (11.3%) cases of purulent otitis media and 26 cases of suppurative mastoid involvement (29%) of the cases of purulent otitis media. This is a very high percentage considering the fact that those cases had been observed from the very start and treated according to the best principles. How much oftener these complications must occur when the ears are not examined at all!

I would like to report a case of the opposite type that is one in which the mastoid symptoms were so pronounced that typhoid fever was not diagnosed until very late:

On October 31, 1903, Miss M. G. L., 18 years old, was sent to me from Palo Alto by Dr. Clelia D. Mosher. The patient had had measles as a baby and could never hear well with the right ear. This had been discharging for the last five months. The right drum showed a large perforation in the posterior lower segment filled out by a polypus. After this was removed a quantity of membranous detritus was taken out with a probe and tincture of iodine introduced on a cotton carrier. After a few treatments the ear became dry.

Toward the end of November the patient had afternoon temperatures—a rise of from ½ to 1 degree. On December 9 she complained about dizziness. The right mastoid process was sensitive to pressure. Dr. Mosher, after a careful examination, could only find the symptoms of a slight upsetting of the digestive apparatus (coated tongue, yellow skin, headaches) attributed to an error in diet. There was no enlargement of the spleen. On December 10 the temperature was 99, pulse 84; the blood count showed 6000 leucocytes; the urine was normal. That day, during my absence, because of the sensitiveness of the mastoid and the dizziness, a colleague opened the mastoid. The mastoid cells were filled with a yellow watery fluid; swabs remained sterile. On my return December 17, the patient was again put under my care. She was feeling well, only complaining of weakness. The evening temperature was 99.7. On December 19 the temperature rose to 101, going higher, the next few days, to 102.9 on December 21. The patient complained about aching of the eyes and was sensitive to the shaking of the bed. The fundus of the eyes was normal. As the symptoms pointed to meningeal irritation the cerebellar fossa was opened on December 21. Fluid blood was found in the sinus. Aching of eyes and sensitiveness to shaking disappeared. On December 24 the temperature rose to 103.6. I opened the middle fossa, but found no pus. The temperature dropped again to 101.2, rising but slightly the next two days. From December 27 to January 3 the temperature rose to 104.4, pulse 116. On January 3 the sinus was opened and fluid blood found.

During the whole illness the bowels were kept open. The patient was very sensitive to calomel, which caused considerable griping pains. The urine remained normal, repeated blood counts showed from 6000 to 7000 leucocytes, swabs from the wound were either sterile or showed staphylococcus growth, As the condition of the wound did not explain the high temperature nor the pulse indicate any meningeal irritation, I suspected some complication and called in consultation Dr. Herbert C. Moffitt, who found enlargement of the liver and spleen, and ordered the widal test, which was positive. Therefore typhoid fever was diagnosed.

Under Dr. Moffitt's care the patient recovered without any further complication.

The mastoid wound was healed with the exception of a small necrotic spot, which required another operation July 7, 1904, leading to an entire cure by August 6, 1904.

Conclusions: The patient probably had typhoid fever before the mastoid operation. When and where the patient contracted the disease could not be ascertained. The sensitiveness of the mastoid might be explained by its being a locus minoris resistentiæ where the toxins created an irritation. I did not see the patient until a week after the mastoid operation. The sterility of the swabs speak against any infection having been present. My second and third operation and the opening of the sinus were certainly unnecessary and useless. Happily, thanks to the aseptic methods, no permanent harm was done. The fact is of some interest that the symptoms of meningeal irritation disappeared after my first operation, and that the temperature dropped considerably after each operation.

This case teaches us the valuable lesson that in a doubtful case of mastoiditis we should make a thorough physical examination, not only of the ear, but of the whole body.

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